EXHIBIT

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October 31, 2025

Dear Mr. Galipo,

Re: Steffon Barber
Medico-Legal Report

Summary of Education, Training and Experience

I completed medical school in 1990 at the University of Nigeria, Enugu, Nigeria. Upon graduating from medical school, I completed a one-year clinical housemanship at the University of Nigeria Teaching Hospital in the fields of Pediatrics, Internal Medicine, General Surgery, Obstetrics, and Gynecology. After housemanship, I worked as an emergency room physician at a university hospital in Nigeria for approximately three years. I sat for and passed my United States Medical Licensing Examinations [USMLE] while I worked as an emergency room physician. I came to the United States in 1994 through a World Health Organization scholarship to become a visiting research scholar for eight months at the Department of Epidemiology, Graduate School of Public Health, University of Washington, Seattle, Washington.

In 1995, I proceeded to the College of Physicians and Surgeons of Columbia University, New York, at Harlem Hospital Center, to complete residency training in Anatomic Pathology and Clinical Pathology. In 1999, I proceeded to the University of Pittsburgh in Pittsburgh, Pennsylvania, to complete residency training in Forensic Pathology and Neuropathology. I hold four board certifications in Anatomic Pathology, Clinical Pathology, Forensic Pathology and Neuropathology. I also hold a Masters in Public Health [MPH] in Epidemiology from the Graduate School of Public Health at the University of Pittsburgh in Pittsburgh, Pennsylvania. I also hold a Masters in Business Administration [MBA] degree from the Tepper School of Business at Carnegie Mellon University in Pittsburgh, Pennsylvania, one of the leading business schools in the world. I am a Certified Physician Executive and an Honorary Fellow of the American Association of Physician Leadership [AAPL]. I also hold a fifth board certification in Medical Management from the AAPL. I am currently licensed to practice Medicine and Surgery in the State of California.

I am currently the President and Medical Director of Bennet Omalu Pathology [BOP], a California medico-legal consulting firm, and a Clinical Professor at the Department of Medical Pathology and Laboratory Medicine, University of California, Davis. In my capacity as the

Medical Director of BOP, I am a consulting Forensic Pathologist and Neuropathologist to many hospitals in central California and to several counties in northern California. There are less than a few dozen practicing Forensic Pathologists-Neuropathologists in the United States who are board-certified in both Forensic Pathology and Neuropathology.

For over 25 years, I have been involved in over 15,000 death and injury investigations in my career as a Forensic Pathologist and Neuropathologist, which began in 1999. I have personally conducted and performed over 13,000 autopsies and death investigations and examined over 15,000 brain tissue specimens. I also perform trauma pattern analysis in both living patients and deceased patients to determine causes and mechanisms of sustenance of injuries and death. I am also involved in the evaluation of living victims of all types of injuries and trauma, including, but not limited to, victims of assault, traumatic falls, industrial and accidental injuries, medical complications and misadventures, rape, child abuse, and sports-related injuries.

I have performed autopsies and examined the medical records, occupational histories, exposure histories, autopsy tissues, and biopsy tissues of hundreds of living and deceased patients who had been occupationally exposed to asbestos, solvents, and other types of toxic agents. I have performed differential diagnosis, made disease diagnosis, and determined medical causation of diseases involving all types of occupational exposures, including asbestos-related diseases and malignancies like malignant mesothelioma.

I have been consulted and retained as an expert witness in 2,000-3,000 cases involving all types of medico-legal cases across all jurisdictions in the United States, including federal, state, county, and municipal courts and arbitration panels, in both civil and criminal cases, for the plaintiff, defense, district attorneys, and public defenders. I have been involved as an expert witness in complex class action and industrial lawsuits involving thousands of individuals and major corporations.

My areas of interest and focus include brain pathophysiology, brain injuries, and brain trauma, in both living and deceased patients. I identified Chronic Traumatic Encephalopathy [CTE] in a retired football player when I performed an autopsy and examined the brain of Mike Webster in 2002. Subsequently, I identified CTE in other high-impact, high-contact sports athletes and in military veterans suffering from Post-Traumatic Stress Disorder [PTSD]. Since 2002, CTE has received international attention from the sports industry, sports medicine, and neuroscience. My work has been featured extensively in all media platforms across the world. My work and life were featured in a major Hollywood film, *Concussion*, released in December 2015 by Sony Motion Pictures, in which the renowned actor, Will Smith, played me as Dr. Omalu. Several New York Times best-selling books have also been published on my life and work, including *The League of Denial* and *Concussion*. I have published several books including my memoir, *Truth Doesn't Have a Side*, which was published in August 2017. My latest book, *Brain Damage in Contact Sports,* was published in February 2018. I have published extensively in the medical and scientific literature, authoring many scientific papers and book chapters.

I have received three honorary PhD degrees from two universities in the United States and from the Royal College of Surgeons of Ireland in recognition of my work and expertise. I have also received numerous awards from across the world in recognition of my work and expertise in both living and deceased patients. I have received the "Distinguished Service Award" from the American Medical Association [AMA], which is the most prestigious award of the AMA. I have

been honored by the United States Congress, and I have appeared on multiple occasions before committees of the United States Congress and committees of State Legislatures across the Unites States, advising them on matters relating to trauma. In 2019 and 2020, I was appointed to the Traumatic Brain Injury Board of the State of California to advise the state on matters relating to traumatic brain injuries.

Since 1999, I have testified as an expert witness in matters relating to all types of injuries and deaths in over 800 court proceedings across the United States. I have attached a copy of my curriculum vitae, which enumerates my body of work and experience in greater detail. The cases I have testified in, beginning in 2009, are enumerated at the end of my curriculum vitae.

Pursuant upon your request, I have reviewed the following materials sent to me on the case of Steffon Barber, Deceased:

1. State of California, County of San Bernardino search warrant and affidavit for Arrowhead Regional Medical Center to obtain hospital admission blood, copies of medical records, doctor's reports, X-Rays and lab results for patient Steffon Barber.
2. Medical records obtained from Arrowhead Regional Medical Center pursuant to the search warrant.
3. Medical records from San Bernardino County Sheriff's Department.
4. Second Amended Complaint.
5. Deposition of Deputy Alfred.
6. Deposition of Steffon Barber.
7. Audio belt recording of Deputy Alfred.

In order to perform and apply a valid differential diagnosis method including but not limited to causation criteria analysis[1], Central Limit Theorem analysis and Clinico-Pathologic Correlation analysis, on this case, I had to review, document, and analyze the materials sent to me on this case in considerable depth and detail. I also visited the patient, Steffon Barber at the Salinas Valley State Prison, spent two hours, interacted with him, and performed a systems review on him. Such differential diagnosis and reviews would form the foundation for my case-specific and general causation opinions in this case.

Brief Summary of the Prevailing Forensic Scenario[1]

Steffon Barber was born on February 12, 1986. On April 27, 2021, at the age of 35 years, he sustained severe traumatic brain injury [TBI] when he was shot in the head by a police officer.

Medical Records from Arrowhead Regional Medical Center
04/28/2021
Steffon Barber was brought in by air transport to the emergency room at 12:22 a.m. with a history of a severe TBI from a gunshot wound to the posterior vertex of the head and parietal-occipital scalp, with cervical spine tenderness. He had a respiratory rate of 16/min with an oxygen saturation of 100 % in room air. He was oriented in person, and had a Glasgow Coma

[1] This section of the report should not be used to establish the facts in this case and is not intended to be used to establish the facts in this case.

Score (GCS) of 13. There was a 3 cm full-thickness laceration on the right side of the vertex with exposed brain tissue. The cervical spine was tender to palpation.

Orbital-facial computed tomography (CT) showed no significant orbital, facial, or mandibular fractures. There were normal sinuses. Cervical spine CT revealed no acute compression fracture or subluxation from C1 through T3.

CT of the head showed the following:
"Findings: There is comminuted fracture of the right parietal skull with extensive bullet and bone fragments tracking centrally into the brain. There is mild bilateral subarachnoid hemorrhage toward the vertex, subdural hemorrhage along the falx and right tentorium. There is no midline shift. Basal cisterns are still visualized. The visualized paranasal sinuses and mastoid air cells are clear. There are no erosive changes in the skull base. The remaining sulci and ventricles are within normal limits. The remaining cerebral parenchyma, brainstem and cerebellum appear to be normal. The superior sagittal and transverse sinuses are within normal limits. There are no definite findings to suggest hyperdense sign in the bilateral middle cerebral and basilar arteries. The ocular globes are intact and symmetric bilaterally.

Impression: Gunshot wound to the right parietal vertex with comminuted fracture and bullet/bone fragments tracking centrally into the brain. Bilateral parietal subarachnoid hemorrhage. Subdural hemorrhage along the falx and right tentorium. No gross herniation at this time."

A computed tomography angiography (CTA)/computed tomography venography (CTV) showed no apparent vascular injury or aneurysm/sinus injury. Electrocardiogram (ECG) revealed sinus bradycardia with marked sinus arrhythmia. There was early repolarization (ST elevation with normally reflected T-wave).

During a neurosurgical review, it was noted that Steffon Barber was confused but was able to move his four extremities. There was an open wound at the right parietal vertex, three centimeters off the midline, with the herniation of brain matter and a blood clot. There was no active bleeding. He was intubated in the trauma bay and continued to remain hemodynamically stable.

The primary impressions were gunshot wound of the head, traumatic encephalopathy and traumatic subarachnoid hemorrhages.

Mr. Barber was subsequently admitted to the surgical intensive care unit (SICU) and was commenced on empirical antibiotics. He was prescribed and administered Keppra and Dilantin for seizure prophylaxis. Continuous elevation of the head of the bed to 30 degrees was recommended.

He underwent a bedside washout, irrigation and debridement of a right open gunshot wound with depressed skull fracture, dural defect and extruding brain. There were necrotic brain tissues and bone fragments with dark blood clotting in an open gunshot wound in the vertex of the right parietal scalp. He also underwent titanium cranioplasty and subgaleal drain placement. There were a 3 cm in length wound with jagged edges, disrupted and frayed galeal edges, comminuted skull fractures, herniating liquified brain tissue and hematoma from the cranial defect, and bleeding from the brain tissue.

04/29/2021
Mr. Barber remained intubated with a GCS of 11T. His postoperative CT showed no new hemorrhage. He followed simple commands in the right upper extremity, such as thumbs up. There was no movement in the left upper extremity or lower extremity. He could withdraw in the right lower extremity. The surgical repair site was covered with a clean and dry dressing. Dr. Li advised a plan for extubation and the use of sequential compression devices (SCDs) for prophylaxis. Physical therapy (PT), occupational therapy (OT), and speech therapy (SP) consults were ordered.

04/30/2021
Mr. Barber's GCS improved to 13. All subgaleal drains were removed, and a staple was placed at the drain exit site. The procedure was well-tolerated, and staple removal was scheduled for the 14th postoperative day.

05/01/2021
Mr. Barber was transferred to the internal medicine team for the continued medical management of his traumatic brain injury. His surgical diagnoses remained a gunshot wound to the head, traumatic subarachnoid hemorrhage, and traumatic encephalopathy.

05/03/2021
Dr. Schiraldi evaluated Mr. Barber and recommended a diagnostic angiogram to identify any potential pseudoaneurysm and address it to prevent future hemorrhage. The benefits, risks, and alternatives were discussed, and Mr. Barber expressed understanding and consented to proceed with the procedure. He was also deemed medically stable for transfer to the Moreno Valley Riverside University Health System (RUHS) Jail ward, and Dilantin therapy was discontinued.

05/04/2021
Mr. Barber was planned for a digital subtraction angiography (DSA).

05/05/2021
The planned DSA was rescheduled for Friday. Mr. Barber's transfer to the Moreno Valley RUHS Jail ward was to take place following completion of the procedure.

05/6/2021
Mr. Barber underwent a diagnostic cerebral angiogram via right femoral artery access, performed by Dr. Schiraldi, which revealed no significant cerebrovascular abnormalities. A physical therapist evaluated him and recommended acute rehabilitation, as he required maximum assistance due to his neurological deficits. Owing to his condition, he was not yet fit to return to jail but was deemed stable for transfer to the TSS team. He was assessed as not requiring antiepileptic medications or antibiotics. Staple removal was scheduled for May 12, 2021, and the right groin bandage was to be removed on May 8, 2021.

05/10/2021 – 5/11/2021
Mr. Barber exhibited generalized weakness and lethargy and was advised to increase the frequency of his physical therapy sessions.

05/12/2021
Mr. Barber reported functional improvement with physical and occupational therapy. He demonstrated increased movement in his right lower extremity and limited motion on the left

side of his body, though he was able to improve mobility during therapy sessions. Sensation was decreased in the left upper extremity (LUE) and both lower extremities (LE). Neurosurgery was consulted for a wound check evaluation.

05/13/2021
Mr. Barber had his staples and drain removed, and the scalp surgical incision was noted to have healed well.

05/14/2021
Mr. Barber had muscle spasms in his left leg overnight and was given oral Robaxin.

05/19/2021
Mr. Barber attended a court hearing and reported that he had slid off his chair but did not experience a fall. He complained of buttock pain, which he attributed to sitting in a wheelchair for an extended period.

05/21/2021
Mr. Barber reported having a headache but declined medication. His symptoms resolved after elevating the head of his bed by 30 degrees.

Records from the San Bernardino County Sheriff-Coroner Department indicate the following diagnoses for Steffon Barber stamped 10/08/2021:
1. Hemiplegia and hemiparesis
2. Personal history of trauma brain injury

Dr. Bennet Omalu's visit and clerkship of Steffon Barber at the Salinas Valley State Prison on October 29, 2025[2]
I visited Steffon Barber at the Salinas Valley State Prison on October 29, 2025. I met with him for two hours beginning at 08:20 a.m. and ending at 10:20 a.m. I performed clinical clerkship and systems review of him by interviewing him and examining him physically. I reviewed his family, social and developmental history.

He was wheelchair bound and could not ambulate independently without the wheelchair. He had significant loss of power and weakness of his upper and lower extremities, accentuated on the left side. He appeared very reserved and subdued, drained and tired. He spoke very softly and became tearful intermittently.

After the shooting, Steffon Barber attempted to get out of bed at the hospital and a nurse told him not to do it, that he was paralyzed and could not walk. His entire left side was paralyzed. He was told that he was shot in the head by a police officer. At this time, he showed me his head, with two large scars on the vertex of the head in the bilateral posterior frontal and parietal scalp and the right temporal scalp.

He was wounded badly and until this day, his left upper and lower extremities were "messed up badly". He could not walk and is dependent on his wheelchair for ambulation.

[2] My interactions and conversations with Steffon Barber were not recorded and could not be recorded given that I met him in a prison setting as an incarcerated inmate.

Steffon stated that he felt disabled. “Everything is so difficult to do”. He experienced constant pain all over his body especially constant stinging and needle-pricking pain in his lower extremities, which never goes away. He has good days and bad days.

He suffered intermittent headaches and has noticed that he was becoming more and more forgetful. He can comprehend stuff, but retaining things and what he has read is becoming a lot harder and difficult. He stated that he felt depressed and humbled. “I am disabled, I cannot do things I used to do, and will like to do. I will like to run. I will like to shoot a hoop or two from time to time. But no, I can no longer do any of that. I am disabled.”

Steffon had wanted to be an underwater welder. He could not swim and was planning to learn how to swim. But now, he cannot do any of that. He cannot learn how to swim and has to forget his dreams of being an underwater welder. “That is what I have always wanted to do”.

Medico-Legal Questions

1. **What were the characteristics and trajectory of the bullet of the gunshot wound Steffon Barber sustained?**
    a. **What was Steffon Barber’s body positioning while he was shot?**
    b. **What injuries or damages were caused to Steffon Barber by the gunshot?**

Medicine is a life science, which is evidence based. The practice of medicine is guided by established standards and generally accepted principles, which certified physicians must adhere to. The specialties and the categories of physicians who are most proficiently trained, specialized, and competent in the accurate determination of the cause, mechanism and manner of death and the mechanisms of sustenance of serious bodily injury and possibly lethal trauma are the forensic pathologists. Steffon Barber suffered serious bodily injuries.

It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of traumas and injuries. Applying the clinico-pathologic method of differential diagnosis, a specific documented pattern of trauma can be evaluated, translated, and applied to the determination of the mechanisms of generation, causation, and sustenance of the specified trauma pattern, with a reasonable degree of medical and scientific certainty; based on the differential diagnosis of the established common knowledge and generally accepted principles of trauma patterns and their mechanisms of generation, causation, and sustenance. The documentation and translation of patterns of trauma can be done by a variety of methods including but not limited to radiological methods, autopsy methods, clinical physical examination methods, and applied clinico-pathologic methods.

The patterns of injuries generated by gunshots, firearms and ballistics weapons, and the mechanisms of generation, causation, and sustenance of these patterns of injuries are very well-established in the medical literature and are common knowledge. Based on the prevailing forensic scenario, and on the generally accepted principles and common knowledge of medicine and science, and based on the global constellation, configurations and anatomic conformations

of the gunshot wound sustained by Steffon Barber, the mechanisms of generation, causation and sustenance of his injuries can be determined with a reasonable degree of medical certainty.

Based on the physical characteristics and physics of ballistics, partially burnt and hot residues of gunpowder and soot travel behind the bullet when it exits the muzzle, and due to gravitational forces and the differential densities of the bullet, soot, and residues of gunpowder in the gravitational field, the bullet can travel longest, followed by the partially burnt gunpowder residues, which travel longer than soot. Soot will travel for about 1 foot, before it is pulled down by gravitational forces, and the partially burnt gunpowder residue will travel for about 2-3 feet before it is pulled down by gravitational forces. Therefore, if the muzzle of the gun were closer to the skin by less than 1 foot, you would expect to find marginal soot deposits around the gunshot wound of entrance [close range shot]. If the muzzle of the gun were closer to the skin by less than 2-3 feet, you would expect to find powder stippling around the gunshot wound of entrance [intermediate range shot]. If the muzzle of the gun were located greater than 2-3 feet away from the skin ad infinitum, you would expect to find only marginal abrasions around the wound without soot deposits or powder stippling [distant range shot]. If there is an eccentric accentuation of the width of the marginal abrasion, it may suggest that the muzzle of the gun was not located perpendicularly to the skin when it was fired but rather located in the direction of the eccentric accentuation of the marginal abrasion.

The direction of travel of a bullet inside the body can be determined in the three planes of nature with the body disposed in the universal anatomic position, by the systematic tracking and description of the anatomic pathway of the bullet, tissue disruptions, damages and injuries, correlated with the anatomic topographic locations of the gunshot wound of entrance, gunshot wound of exit and recovery of the bullet, or where the bullet settled.

Based on these common knowledge and generally accepted principles of medicine and science, Steffon Barber sustained a gunshot wound of his head, with the entrance wound located in the vertex of the head in the right medial and posterior frontal and parietal scalp. The anatomic location of the gunshot wound of entrance indicates that at the time he was shot, Steffon Barber's head was located at a vertical level that was lower than the vertical level of the gun that fired the bullet. Steffon Barber was not standing erect on his feet when he was shot. The prevailing forensic scenario suggests that Steffon Barber was sitting down in a car when he was shot. This scenario would be consistent with the anatomic location of the gunshot wound of entrance in the vertex of the head.

The bullet perforated, contused and lacerated the dorsal right posterior frontal and parietal scalp, perforated and fractured the frontal and parietal calvarium, perforated, contused and lacerated the dorsal dura mater and meninges, perforated, contused and lacerated the right cerebral hemisphere where the bullet fragments came to settle. There were extensive contusions and lacerations of the right dorsal cerebral hemisphere with parenchymal pulpifaction, subdural and subarachnoidal hemorrhages. Steffon Barber suffered severe traumatic brain injury caused by the gunshot wound and bullet.

He suffered both focal and diffuse, primary and secondary severe traumatic brain injury with a cavitatory effect given that the cranial cavity is an enclosed space, and a bullet penetrates this enclosed space at very high velocities of greater than 1200 feet per second, and transfers large amounts of kinetic energy into the intra-cranial cavity and brain.

The bullet traveled in a forward, downward and rightward trajectory into the brain. The anatomic locations, configurations and conformations of the gunshot wound are consistent with the shooter and gun located at back of Steffon Barber's head when the bullet was fired. Steffon Barber was not facing the officer who shot him when the bullet was fired.

The clinical documentations and descriptions of the gunshot wound of entrance on Steffon Barber's head did not mention any soot deposits or powder stippling. This would mean that Steffon Barber suffered a distant shot. He was not close to the officer who fired the bullet. He was located at a distance that was far removed from the muzzle of the gun, at greater than 2-3 feet ad infinitum. The gunshot wound Steffon Barber suffered was not a contact gunshot wound, it was not a loose contact or close range gunshot wound. It was not an intermediate range gunshot wound. It was a distant range gunshot wound.

The global configurations and conformations of the gunshot wound of the head that Steffon Barber sustained are consistent with the stated scenario in Steffon Barber's deposition that Steffon Barber did not see the officer who shot him, he did not know that it was an officer and had his back facing the officer when he was shot. He was not charging at the officer with his car or attempting to assault the officer with his car. He was not facing the officer who shot him, when he was shot. He was sitting in the seat of a car when he was shot by an officer who was standing on his feet. The vertical level or height of the muzzle of the gun was either higher or about the same vertical height as the level of his head when the bullet was fired. The bullet entered the head from the top of the head, and in the back of the head.

2. On April 27, 2021, Steffon Barber sustained severe traumatic brain injury when he was shot in the head by a police officer. He was diagnosed with a gunshot wound of the head and traumatic encephalopathy at the hospital.
   a. Did the severe TBI cause permanent brain damage?
   b. What are the expected long term effects, outcomes and sequelae of his severe TBI?
   c. Does the severe TBI he suffered independently, significantly and substantially increase his risk of developing neurodegenerative diseases including but not limited to all types of dementias, traumatic encephalopathy and chronic cerebrovascular disease?

As has been presented above, yes, Steffon Barber suffered severe TBI on April 27, 2021 when he was shot in the head by a police officer. The severe TBI he sustained from a gunshot wound of his head is an independent, significant and substantial contributory, aggravating and accelerating factor for neurodegenerative diseases including but not limited to dementias, traumatic encephalopathy and chronic cerebrovascular diseases.

The differential diagnosis of his medical history, which has been summarized above confirms that the severe TBI and gunshot wound of the head he was diagnosed with caused substantial permanent, progressive and cumulative brain damage and serious bodily injury that is consistent with traumatic encephalopathy, a neurodegenerative disease and a type of dementia, which will progress as time goes on and as he gets older into more advanced forms of dementias and neurodegenerative diseases including Alzheimer's Disease pathological changes in the brain

All forms of chronic brain damage and neurodegeneration following TBI, traumatic encephalopathy and all forms of dementia belong to one and the same spectrum of diseases or dementias[2].

The human brain is a post-mitotic organ and as a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself following injury and trauma-induced cellular damage. This means that when the human brain suffers any type of irreversible injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy, and as time progresses the permanent brain injury may progress into a neurodegenerative disease. All forms of traumatic injuries to the brain, including the milder forms of TBI like a concussion are permanent brain injuries which can be progressive with time[3].

A history of exposure to single, episodic, or repetitive TBI is reliable differential diagnosis evidence for the reasonable determination of permanent brain damage and the expected outcomes and sequelae of TBI. This means that physicians reasonably rely on the patient's medical, social, educational, and occupational histories as narrated by the patient, family members, friends and colleagues to perform this differential diagnosis. A calculation of the quantitative threshold dose of exposure to TBI has never been required for the valid differential diagnosis of TBI outcomes and sequelae, and to link a patient's TBI exposure to these outcomes and sequelae. It is an accepted methodology across health organizations and specialties to use exposure history for the differential diagnosis of injury outcomes across all diseases and disease syndromes. This is an established standard of practice.

Medicine is a life science, which is evidence based. The practice of medicine is guided by established standards and generally accepted principles, which certified physicians must adhere to. Differential Diagnosis and trauma pattern recognition, interpretation and analyses are the fundamental methodologies physicians adopt in the determination of expected and anticipated outcomes and sequelae of disease and trauma. It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of injuries, outcomes, and sequalae.

After I described traumatic encephalopathy in the brains of football players in 2002 in Pittsburgh, Pennsylvania I performed and applied the method of differential diagnosis and the Bradford Hill Criteria[1,4] to determine the contributory and causal risk factors for TBI and traumatic encephalopathy. Traumatic encephalopathy was defined as the "disturbance of structure and/or function of nerve cells, glia, or intracranial vessels resulting from injury"[5]. Such a disturbance is expected to manifest with a constellation of syndromic signs and symptoms with time and over time following traumatic brain injury. I performed an extensive review of the medical literature for traumatic brain injury and traumatic encephalopathy beginning with Hippocrates who first recognized and named concussions at about 400 B.C.[6,7], followed by Claudius Galenus in the 2nd century AD, who both named concussions commotio cerebri[6,7].

In my continuing differential diagnosis review and method, which has spanned across the centuries from about 400 B.C. to 2022, I was able to determine that traumatic encephalopathy was not a novel or new disease, and that traumatic encephalopathy has been a disease that was very well-recognized and accepted by doctors across the world for centuries[7]. The disease traumatic encephalopathy had been known by a variety of names across the centuries, one and same disease, but different labels, syntax and names, given by different doctors, researchers and agencies[7,8] across the world and global scientific community. In 2014, I published a paper and

book chapter titled "Chronic Traumatic Encephalopathy"[8] in which I listed the names I had identified that traumatic encephalopathy had been known by over the centuries. The table is presented below, copied from page 40 of the article:

Table 1. Syntactics and semantics of CTE across the centuries

1. Cerebral neurasthenia
2. Chronic postconcussion syndrome
3. Chronic TBI/chronic brain injury
4. Compensation hysteria
5. Concussion neurosis
6. Delayed traumatic apoplexy
7. Dementia pugilistica
8. Dementia traumatica
9. Encephalopathia traumatica
10. Litigation neurosis
11. Postconcussion neurosis
12. Postconcussion syndrome
13. Posttraumatic concussion state
14. Posttraumatic dementia
15. Posttraumatic head syndrome
16. Posttraumatic parkinsonism
17. Posttraumatic psychoneurosis
18. Posttraumatic stress disorder
19. Punch drunk/punch-drunk state
20. Terror neurosis
21. Traumatic constitution
22. Traumatic encephalitis
23. Traumatic encephalopathy
24. Traumatic encephalopathy of boxers
25. Traumatic hysterias
26. Traumatic insanity
27. Traumatic neurosis
28. Traumatic psychosis

In 1964, the United States Congress of Neurological Surgeons formed an Ad Hoc Committee to study head injury nomenclature, and their report was published in 1966 titled *Proceedings of the Congress of Neurological Surgeons in 1964: Report of the Ad Hoc Committee to Study Head Injury Nomenclature*[5]. In this report United States neurosurgeons identified and described traumatic encephalopathy as "Disturbance of structure and/or function of nerve cells, glia, or intracranial vessels resulting from injury."[5]

The differential diagnosis method is a very well established and generally accepted methodology in the medical sciences and is common knowledge for the determination of disease outcomes. The generally accepted principles and standards of practice of the differential diagnosis of TBI and the sequelae of TBI are based on the qualitative/quantitative history of exposure to TBI like Steffon Barber was exposed to on April 27, 2021.

The type of gunshot wounds that Steffon Barber suffered April 27, 2021 are commonly known to cause TBI and permanent brain damage[9-14]. Such a violent high-velocity injury causes the transfer of linear/ translational and angular/ rotational acceleration-deceleration forces and kinetic energy to the skull and brain, which is known to cause primary and secondary TBI with cerebral edema, brain swelling and herniation, cerebral hypoperfusion and raised intracranial pressure, axonal shearing, focal and diffuse traumatic axonal and micro-vascular brain injury, neuropil contusional hemorrhages and necrosis, parenchymal lacerations and permanent brain damage[11-17].

In this case of Steffon Barber, he began manifesting the symptoms and signs of TBI immediately after he was shot. He manifested, continues to manifest, and is suffering from the symptoms and signs of TBI, TBI outcomes and sequelae, neurodegenerative disease and dementia, as have been clearly demonstrated by his prevailing medical history, which has been summarized above. In addition to the signs and symptoms of acute mild, moderate and severe TBI, exposure to TBI may present immediately or after a delayed interval with post-concussion syndrome, post-traumatic epilepsy, post-traumatic encephalopathy [PTE], mood disorders, behavioral disorders, mild cognitive impairment, neuropsychiatric disorders including drug abuse and alcoholism, motor disorders including but not limited to cerebellar ataxia, motor neuron disease and Parkinson's disease, dementia including Alzheimer's Disease, CTE, Post-Traumatic Stress Disorder, neurovascular diseases, somatic and neurosensory symptoms like headaches, body aches and pain, and increased risk of suicide[8,18-71]

While TBI has been confirmed to be a causal risk factor for developing various dementias and neurodegenerative diseases as has been stated above, dementias like Lewy Body Disease/ Dementia [Parkinson's Disease] have exhibited one of the strongest and most compelling link to TBI as a causal risk factor amongst other spectrum of dementias and neurodegenerative diseases[25,49,52-57].

For a patient like Steffon Barber, the expected outcomes of a disease or injury can be further analyzed and assessed epidemiologically using published epidemiological studies founded upon previously established and generally accepted principles of medicine and science. As an epidemiologist, I would apply many published long-term population-based epidemiological studies to the case analysis of Steffon Barber's expected TBI risk outcomes and sequelae.

Exposure to all forms of TBI is associated with elevated risks of impaired adult functioning across all outcome measures[39] in a patient like Steffon Barber. Exposure to traumatic brain injury may be associated with and is an independent risk factor for premature deaths at younger ages, with approximately 24.3 years of life lost[38,39,72,73] and unadjusted odds ratio of 3.6 for premature death among traumatic brain injury patients, with increased risks for all causes of premature deaths, and even higher absolute rates of death in patients with co-mobidities[38,39].

There is a 400-fold increased life-time diagnosis of substance abuse in patients with a history of traumatic brain injury[38,39]. There is a dose-response relationship with outcome measures and injury severity with recurrent or repetitive traumatic brain injury associated with higher risks sometimes up to 3-fold increased risk compared to single-episode traumatic brain injury[38,39] observed with injury severity.

The human brain is a post-mitotic organ and as a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself following injury and trauma-induced cellular damage. This means that when the human brain suffers any type of significant or irreversible

injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy, and as time progresses the permanent brain injury may progress into a neurodegenerative disease.
Mild TBIs and concussions can aggravate dementia in a patient already suffering from dementia. Even a single episode of a mild TBI or a concussion is associated with an increased risk of cognitive decline and dementia. This association is, however, more pronounced with more severe TBIs or repeated mild TBIs[24,74]. A history of TBI may cause cognitive impairment to appear two or more years earlier than it would otherwise, and is a substantial risk factor for cognitive decline in older adults with the beginning of moderate cognitive impairment and Alzheimer's Dementia[75].

In a nationwide population-based observational cohort study in Denmark, citizens data from national registries of all people born in Denmark who were living in the country on January 1, 1995, and who were at least 50 years old at some point during follow-up, 1999-2013, were studied. Information on TBI was obtained from the Danish National Patient Register [NPR]. Information on dementia was obtained from by combining data recorded in the NPR, the Danish Psychiatric Central Register, and the Danish National Prescription Registry. The long term risk of dementia after TBI was established using survival analysis. Data for a cohort of 2.8 million people were used for a total of 28 million person years at risk of dementia. 132 thousand individuals [4.7%] had at least one TBI, and 127 thousand [4.5%] had incident dementia during the study period. The risk of dementia was highest within the first six months after TBI, and also increased with increasing number of events. TBI was associated with an increased risk of dementia both compared with people without a history of TBI and people with non-TBI trauma. The overall risk of dementia in individuals with a history of TBI was 24 percent higher than those without a history of TBI. A single episode of severe TBI increased the risk by 35 percent. A single episode of mild TBI or concussion increased the risk by 17 percent. Even a single episode of mild TBI was associated with a significantly higher risk of dementia[24].

"Dementia-related manifestations after TBI include abnormalities of memory, thinking and concentration, communication, interactions with others, mood, and personality. TBI may cause rapid, complex structural, and physiological changes in the brain, that in addition to the released biomarkers, subsequently lead to an abrupt coping crisis and abnormal responses like excessive anxiety and depression. This disorder might happen when symptoms from psychological trauma disrupt daily functioning for at least a month. A study showed that during this time, subjects who sustained TBI were 4–6 times as likely to develop dementia than those without TBI. Furthermore, a concussion (mild TBI) or other TBI can increase the risk of developing dementia even after 30 years of the primary insult"[76].

"There was a significant interaction between TBI severity and age category such that moderate/severe TBI was associated with increased dementia risk across all ages, while mild TBI became a relatively more important dementia predictor with increasing age" in the elderly[77].

"Overall, TBI has been reported to confer a 1.6- to 3.7-fold increased risk of dementia… Furthermore, TBI may impact cognitive function to a different degree depending on the age of onset. Previous research has suggested that slow cognitive decline may occur after a TBI at any age though this decline appears to be more severe among individuals who experience a TBI at an older versus younger age. This may be related to brain plasticity. Older individuals may have less ability to compensate for TBI-related brain damage during the initial recovery period or may experience greater brain degeneration after the initial recovery period due to reduced plasticity of the aging brain. Therefore, when exploring the relationship between TBI and dementia, the timing

of TBI over the life course deserves more attention...[78] The odds ratio of dementia was 1.27 for TBI at any age, 1.55 for TBI at 50 to 59 years, and 1.67 for TBI at 60 to 69 years. TBI across all ages including the elderly is associated with an increased risk of dementia[78].
Steffon Barber exhibited novel and progressive signs and symptoms of TBI and permanent brain damage following his April 27, 2021 gunshot wound of the head. In an epidemiological population-based study by Nordstrom A and Nordstrom P, it was confirmed that the risk of a dementia or neurocognitive disorder diagnosis following TBI was highest during the first year after the TBI[79]. In this study all individuals aged 50 years old and older on December 31, 2005, were included for a total of 3.3 million persons. The diagnosis of dementia and TBI were tracked through nationwide databases for seven years until December 31, 2012. Individuals diagnosed with TBI were matched with up to two controls, and individuals diagnosed with dementia were matched with up to two controls. The mean follow-up period was 15.3 years. 6.3% of the participants with TBI were diagnosed with dementia with an adjusted odds ratio of 1.81. The association between TBI and dementia was strongest in the first year after TBI, but the risk remained significantly greater than 30 years after TBI. The history of TBI may accelerate the age of onset of cognitive impairment by two or more years[75].

The case of Steffon Barber is not an outlier and is not an anomaly. He was a representative index of TBI in human beings, as a member of the human race. The differential diagnosis method identifies and confirms that exposure to TBI especially blunt force trauma of the head in any form of human activity is the single most important risk factor for traumatic encephalopathy and dementia[7]. Over 2000 years after the work of Hippocrates in identifying concussions, Ling H et al. at the Institute of Neurology, University of London have reported that about 12% of the general population suffer from traumatic encephalopathy due to exposure to TBI[80]. Steffon Barber is one of these 12%.

A young patient like Steffon Barber who sustained severe TBI at the age of 35-years-old, more likely than not, possesses a significantly and substantially increased risk of developing Young-Onset Dementia [YOD], which is defined by the onset of dementia before the age of 65-years-old[81]. There is a strong association between YOD of non-AD forms and TBIs of different severity. In a national wide cohort study Nordstrom P et al[81] studied a cohort of about 810,000 Swedish men with a mean age of 18 years old, who were conscripted for military service between 1969 and 1986. TBIs, dementias and covariates were extracted from national registers. Time-dependent exposures were evaluated using Cox proportional hazard regression models. There was a median follow-up of 33 years. One severe TBI like we have in this case was associated with a high hazard ratio of 11.4 for YOD of non-AD forms[81].

In their paper titled “Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias”, Plassman BL et al[82] reported their findings in a population-based prospective historical cohort study. The study included men who were world war II navy and marine veterans in 1944 to 1945 who were hospitalized during their military service with a diagnosis of either a non-penetrating head injury or another unrelated condition. The entire sample was then evaluated in 1996 to 1997 for dementia and AD using a multistage procedure. There were 548 veterans with head injury and 1228 veterans without head injury who completed all assigned stages of the study. Both moderate and severe TBI were associated with significantly increased risks of dementia and AD with hazard rations of 2.32 and 4.5, respectively. They concluded that moderate and severe head injuries in early adult life were associated with increased risk of AD and other dementias in late life. The risk of dementia and AD increased with the severity of the TBI[82].

Using the Swedish Twin Registry, over 35,000 dementia-free twins were followed up for up to 18 years and TBI history was identified using medical records. In a multi-adjusted generalized estimating equation model, there was an odds ratio of 1.03 to 1.57 for developing dementia following TBI at any age; and odds ratio of 1.12 to 2.49 for TBI at 50-59 years old, and an odds ratio of 1.12 to 2.49 for TBI at 60-69 years old. Zhang L et al[78] concluded that TBI at any age but especially between the ages of 50-69 years old, was associated with significantly and substantially increased risks of developing dementia[78].

In yet another nationwide population based study, Yang JR et al[34] used Taiwan's National health Insurance Research Database to identify 501,889 adults who had ≥1 medical record of craniofacial trauma between 2000 and 2010 and did not have a dementia diagnosis at baseline. Diagnosis of craniofacial trauma including facial bone fracture and TBI, and dementia were made using ICD-9 codes. The standardized incidence ratio was used to determine whether craniofacial trauma was associated with a greater risk of incident dementia compared with the general population. The Cox proportional hazards model was used to predict the risk of dementia among the trauma cohort by comparing patients with and without comorbidities. Out of 501,889 patient with craniofacial trauma, 7804 [1.5%] developed dementia. Facial bone fracture was shown to be associated with an increased dementia risk compared with the general population. Craniofacial trauma accompanied with postinjury comorbidities was associated with an increased risk of dementia during follow-up periods compared with the group without comorbidities. Yang JR et al concluded that craniofacial traumas were significantly and substantially associated with an increased risk of subsequent dementia[34].

In order to assess the association between TBI and dementia, Simmonds, E et al[83] performed a population-based study using Welsh [UK] electronic health records, which included 20 years of data and 1.7 million individuals with hospital or general practitioner diagnoses of dementia and TBI. Study participants were between the ages of 30 and 65 years old in 1999 without a previous dementia diagnosis. The long-term risk of dementia after TBI was established using Cox proportional hazard models. There were about 43 thousands individuals with dementia, 10,164 individuals with a history of TBI, and 1.7 million controls. TBI was associated with increased risk of dementia including vascular dementia, unspecified dementia and AD[83].

Iacono D et al[84] concluded that a previous TBI exposure was a significant age-lowering factor for the onset of cognitive decline in either Alzheimer's Disease or non-Alzheimer's Disease conditions independent of sex, race, attained education or clinical diagnosis. Previous TBI exposures accelerated the onset of later cognitive decline across different brain diseases, with or without dementia. They analyzed data from the National Alzheimer's Coordinating Center [NACC] which provide information on history of TBI and longitudinal data on cognitive and non-cognitive domains for each available subject. They examined a total of 609 NACC subjects with a documented history of previous TBI of any type. They compared subject with or without a history of previous TBI at the time of their first cognitive decline assessment. The mean age of TBI-positive subjects was lower than the mean age of TBI-negative subjects at the time of their first cognitive decline assessment. In addition, neuropsychiatric and neurobehavioral symptoms were much more frequent in TBI-positive vs TBI-negative subjects.

In the Sariaslan et al paper[39], they identified and studied 1.3 million Swedish children born between 1973 and 1985 [12 years] for 41 years, and excluded children who could not be linked to both of their biological parents, who had died, or had migrated before the age of 26 years old, or

lacked data on parental sociodemographic factors, and adult outcome measures. The final sample was 1.1 million children. The study TBI patient was defined as an individual who had sought treatment for at least one episode of concussion [mild TBI] or moderate-to-severe TBI before 25 years as a TBI patient. The following outcomes and sequelae were studied: disability pension, specialist diagnoses of psychiatric disorders and psychiatric inpatient hospitalization, pre-mature mortality [defined as death before the age of 41 years old], low education attainment [defined as not having achieved secondary school qualifications] and receiving means-tested welfare benefits. There were 104,290 individuals who sustained TBI before the age of 25 years old. 77% of these patients, an overwhelming majority, suffered from mild TBI. TBI was associated with elevated risk of impaired adult functioning across all outcome measures. TBI contributed an absolute risk of 10% for specialist diagnoses of psychiatric disorders, low educational attainment, and welfare recipiency, and approximately 6% for disability pension. TBI patients were nearly 60% more likely to be hospitalized for any psychiatric disorder. Social functioning impairments were also 60% more likely in TBI patients than in unrelated controls; and 28% more likely to have attained a low level of education in adulthood, as well as receiving welfare benefits. Interestingly, these findings were not and could not be attributed to pre-existing or contemporaneous co-morbidities like neurological conditions and psychiatric disorders. The effects of TBI on the outcomes did not change materially over time. The age at first TBI, importantly, was a very strong moderator of poor functioning in adulthood with a positive association between older age at first TBI and all outcomes. Steffon Barber's previously attained intellectual and executive functioning has been significantly impaired. He can no longer become the underwater welder or anything similar that he had wanted to become. He is even expected to progressively deteriorate intellectually as time passes.

In the Fazel et al paper[38], they performed a 40-year study of 218,300 TBI patients and control-matched them with about 2.1 million members of the general population and 150,513 patient siblings without TBI. The patients had at least one patient episode of primary, secondary, or additional diagnoses. They excluded patients who died within 6 months of diagnoses of TBI to exclude immediate causes of death that could have caused the TBI. Compared to the general population, the unadjusted odds ratio of premature death among TBI patients was 3.6. Risks for all causes of premature death were elevated, with the largest causes from external factors including injuries, suicide, and assault. There were increased rates of psychiatric disorders in TBI patients including alcohol and drug use disorders and depression. The adjusted odds ratio for premature mortality in patients with both TBI and psychiatric comorbidity, specifically substance abuse and depression ranged from 8 to 24. Suicides in TBI patients like Steffon Barber had an odds ratio of 3.3 when compared to the general population.

In the Madsen et al paper[40] they studied nationwide registers in Denmark covering 7.4 million individuals from 1980 to 2014 and identified 567,823 TBI patients with a concussion [mild TBI], skull fracture or severe TBI [head injuries with evidence of structural brain injury]. Suicides recorded in the Danish Cause of Death register were identified. An overwhelming majority of the TBI patients, 423,502 were diagnosed with concussion [mild TBI]. Among the 34,529 suicides identified, 3536 [10.2%] had been diagnosed with TBI, including 2701 with concussion [mild TBI], 174 with skull fracture and 661 with severe TBI. The suicide rate for the general population was 19.9 per 100,000 person years; the suicide rate for TBI patients was 40.6 per 100,000 person years; the suicide rate for concussion [mild TBI] patients was 38.6 per 100,000 person years. The incidence rate ratio of suicide in TBI patients compared to the general population ranged from 2.64 to 3.35 for the basic research model. In their nation-wide cohort study, they confirmed that TBI patients have a significantly increased risk of suicides when compared to the general

population. Steffon Barber is more likely than not to exhibit remarkably increased risks of mood and psychiatric disorders including self-destructive behavior.

Steffon Barber stands an established risk of socio-economic deterioration and eventual homelessness following his TBI. This is an expected and known outcome of TBI[85-87]. The rates of TBI are significantly higher among individuals experiencing homelessness compared to the general population[86]. In a cross-sectional study design, purposive sampling was utilized to interview 115 English-speaking adults [ages 18-73] in two Colorado cities and 71% of total participants reported a significant history of TBI, and of these 74% reported a TBI prior to experiencing homelessness. Logistic regression models revealed a significant relationship between mental health and acquiring a TBI prior to experiencing homelessness[86].

In a systematic review and meta-analysis paper by Stubbs JL et al[85], they searched without date restrictions for original research studies in English that reported data on the prevalence or incidence of TBI, or the association between TBI and one or more health-related or function-related outcome measures. Studies were included whether they had a group or clearly identifiable subgroup of individuals who were homeless, marginally housed, or seeking services for homeless people. Of the 463 potentially eligible studies identified by the search, 38 studies were included in the systematic review and 26 studies were included in the meta-analysis. The lifetime prevalence of any severity of TBI in homeless and marginally housed individuals was 53.4% [95% CI]. TBI was consistently associated with poorer self-reported physical and mental health, higher suicidality and suicide risk, memory concerns, and increased health service use and criminal justice system involvement[85].

Hwang S et al[87] determined the lifetime prevalence of TBI and its association with current health conditions in a representative sample of homeless people in Toronto, Ontario. 601 men and 303 women at homeless shelters and meal programs were surveyed. TBI was defined as any self-reported head injury that left the person dazed, confused, disoriented or unconscious. The lifetime prevalence of TBI among homeless participants was 53% for any traumatic brain injury. For 70% of the respondents, their first TBI occurred before the onset of homelessness[87].

Having applied and analyzed these general causation principles which the case specific causation principles are based upon, Steffon Barber manifested novel, aggravating and progressive symptoms of brain damage, traumatic encephalopathy and dementia after his TBI. It is common knowledge that traumatic encephalopathy of all types can manifest with symptoms immediately after the occurrence of TBI, or after several hours, several days, several months, several years or several decades after the TBI. Steffon Barber's post-traumatic brain damage and dementia are permanent and are expected to be progressive as time goes by. His symptoms are permanent because TBI is a permanent injury given that the human brain is a post-mitotic organ and every injury to the brain is permanent. It is likely that Steffon Barber will remain wheelchair bound for the rest of his life. He is expected to develop the sequelae of significantly limited mobility like decubitus ulcers, urosepsis and sepsis.

The principles of the central limit theorem [CLT], which guide every measurable human index in science, mathematics, and statistics, dictate that there is a normal broad variation in the manifestations and symptomatic presentations of every disease including TBI. Every patient does not present exactly the same way. About 68%, 95% and 99.7% of all patients would present with indices that are within +/- 1, 2 and 3 standard deviations of the expectation, respectively. Therefore, if an interested party may claim that the manifestations and symptomatic

presentations of Steffon Barber may not be classical manifestations or presentations, this claim may not be scientifically valid since his manifestations and presentations would normally fall within +/- 1, 2 or 3 standard deviations from the expectation.
Steffon Barber has suffered from, and continues to suffer from multi-domain mood, cognitive, intellectual, behavioral, social, motor and somatic impairments pathognomonic of traumatic encephalopathy and dementia. Again, according to the CLT, each aspect of the multi-domain sequelae of TBI, as has been stated above, has been causally associated with exposure to TBI with a continuum and spectrum of causation risk. For example, while TBI has been confirmed to be a causal risk factor for developing various dementia and neurodegenerative diseases as has been stated above, Parkinson's Disease has exhibited one of the strongest and most compelling link to TBI as a causal risk factor amongst other spectrum of dementias and neurodegenerative diseases[25,49,52-57].

The two variables in this instance are TBI, the instigating, causal or risk factor, and persistent post-traumatic syndromes, traumatic encephalopathy and dementia, the outcome. The causal link between these two variables can be established based on the nine Hill's criteria for determining case specific causation[1,4]. The strength of the causal association between TBI and traumatic encephalopathy is very strong, which has been stated above. The epidemiological data linking these two variables are very consistent. The traumatic encephalopathy is temporally related to the TBI, in that the symptoms manifested after the TBI had occurred or where aggravated by the TBI. The biological gradient of TBI and TBI outcomes is well established in that the greater the exposure, the greater the likelihood of the sequelae[37,48,50,88-91]. The link between Steffon Barber's TBI and traumatic encephalopathy is scientifically plausible, reasonable and probable, given that the pathophysiological mechanisms of developing traumatic encephalopathy and dementia following TBI is well known. The cause-and-effect interpretation of TBI and traumatic encephalopathy and dementia does not seriously conflict and is coherent with the generally known facts of the history and biology of traumatic encephalopathy and dementia. There are both epidemiological and experimental evidence as has been presented above that TBI can be a causal, contributory, aggravating and accelerating risk factor for traumatic encephalopathy and dementia. Finally, there are other analogous cause and effect links between TBI and other diseases outside traumatic encephalopathy and dementia. Therefore, the case specific causation in the case of Steffon Barber is consistent with the general causation, and it can be concluded that the severe TBI he sustained from a gunshot wound of the head on April 27, 2021, was a single, independent, substantial, and significant contributory, aggravating and accelerating risk factor for his development of impaired motor functioning and hemiplegia, traumatic encephalopathy and dementia. It does not have to be the only mutually exclusive factor, but a significant and substantial risk factor by itself.

In the case specific causation analysis of Steffon Barber, we may consider the concept of co-morbidity in the medical sciences. For every disease, there are extenuating and aggravating factors, which can either decrease or increase the risk of suffering from or dying from a disease. A contemporaneous or co-morbid disease or factor that increases the risk of a second disease or factor does not denote causation, rather it denotes co-morbidity. Disease or event "A" that is co-morbid with disease or event "B" does not mean disease "A" causes disease "B" and vice versa.

Therefore, any assumed or presumed pre-morbid or contemporaneous occurrence of any other possible disease Steffon Barber may be suspected or alleged to have should be considered as a co-morbidity and not the cause of persistent and progressive symptoms of TBI, traumatic encephalopathy or dementia. This co-morbidity may have increased or decreased the risk of his

traumatic encephalopathy and dementia or may have synergistically and cumulatively aggravated his traumatic encephalopathy and dementia, but it did not cause or initiate his traumatic encephalopathy and dementia.
In conclusion therefore, yes, Steffon Barber suffered severe TBI on April 27, 2021 when he was shot in the head by a police officer. The TBI he sustained from this event at the age of 35-years-old is an independent, significant and substantial contributory, aggravating and accelerating factor for his neurodegenerative disease, dementia and traumatic encephalopathy.

Steffon Barber is already suffering from the signs and symptoms of a neurodegenerative disease following TBI- traumatic encephalopathy and dementia. His disease is permanent and progressive and is expected to progress to advanced dementia across decades. He is at a significantly and substantially increased risk of suffering from advanced young onset dementia [YOD] before the age of 65 years old within 20-30 years from the date of sustenance of severe TBI. He was 35 years old when he suffered severe TBI, therefore he is epidemiologically expected to develop severe and advanced dementia beginning at about 55 years old until about 65 years old. As he gets closer to 55 years old he is expected to need remarkably increasing levels of assistance with activities of daily living, medical attendance and specialized medical care. Beginning after the age of 55 years old up until about 65 years old, he is expected to become fully dependent on specialized assistance with activities of daily living and would need and become dependent on daily and full-time [24/7] basis on specialized medical attendance and medical care until the time of his death[78,79,81,92]. He is expected to remain wheelchair bound with increasingly progressive limitations and impairments of mobility for the rest of his life since the brain damage he has suffered is permanent and progressive as he gets older. He is also expected to suffer and manifest the commonly known sequelae of hemiplegia and impaired mobility like decubitus ulcers, urosepsis and sepsis.

3. Did Steffon Barber experience pain and suffering as a result of his severe TBI and gunshot wound of the head, and for how long?

It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of traumas and injuries and outcomes of traumas and injuries. The patterns of injuries generated by gunshots and all types of ballistics and the mechanisms of sustenance of these patterns of injuries are very well-established in the medical literature and are common knowledge. A specified prevailing pattern of trauma can reasonably predict the mechanisms of sustenance of the prevailing trauma.

Steffon Barber suffered severe TBI as a result of a gunshot wound of the head when he was shot by a police officer. Based on the prevailing forensic scenario, and on the generally accepted principles and common knowledge of medicine and science, and based on the global constellation, configurations, and anatomic conformations of the traumas sustained by Steffon Barber, he experienced pain and suffering beginning from the time of sustenance of his severe TBI and will continue to experience pain and suffering until his death. All forms of TBI, especially severe TBI, the type of TBI suffered by Steffon Barber, are permanent and progressive injuries.

Pathophysiology of conscious pain and suffering

Conscious pain and suffering are initiated by widespread free nerve endings situated in the skin, soft tissues, and organs. Pain can be elicited by multiple types of stimuli classified into three broad categories: mechanical, thermal, and chemical pain stimuli. Nerve endings for pain sensations generate electrical action potentials following all forms of tissue damage caused by all types of energies including, but not limited to, kinetic and mechanical energy from gunshots.

Action potentials are the sub-cellular physiologic basis for noxious conscious sensations and originate from voltage gated sodium and potassium electrolyte membrane pumps in the cell membranes of nerve cells, fibers, and synapses.

It takes few 10, 000th's of a second to generate action potentials. Action potentials are transmitted through nerve fibers to the brain. They are transmitted in peripheral nerves in the Aδ and C fibers for fast and slow pain respectively at impulse rates of 5-30 meters per second and 0.5-2 meters per second, respectively. There is therefore a double pain sensation, a fast-sharp pain, and a slow pain. The sharp pain apprises the person rapidly of imminent danger and prompts the person to react immediately and remove himself from the painful stimulus or imminent danger. The slow pain becomes greater as time passes resulting in continued intolerable pain and suffering prompting the person to continue to try to relieve the cause of the pain and flee from the imminent danger.

As an average human adult, experienced all types of gunshot and ballistics induced pain within milliseconds of contact and penetration of the bullet. One millisecond is one second divided into 1000 parts. For the slowest nervous mechanisms of pain sensation and consciousness, an adult male, like Steffon Barber, felt pain within 100 milliseconds.

Nerve pathways transmitting pain, terminate in the spinal cord. Secondary pathways transmit the pain from the spinal cord to the brainstem and thalamus, especially to the reticular activating system of the brainstem. From the thalamus, tertiary pathways transmit pain to other basal ganglia, limbic cortex, and neocortex of the brain. Pain stimuli are transmitted to the reticular nuclei of the midbrain, pons, and medulla; to the tectal midbrain and the periaqueductal gray matter. These lower regions of the brain, i.e., brainstem, are vital for the appreciation of the suffering types of pain.

Animals with their brains sectioned above the midbrain, to block any impulse reaching the neocortex and cerebral hemispheres, still experience suffering from pain caused by all types of trauma. Complete removal or disconnection of the somatosensory regions of the cerebral hemispheres, like we may have in quadriplegic patients, does not preclude a human or animal's ability to perceive and experience pain.

Pain impulses entering the spinal cord, brainstem and lower centers of the human brain can cause perception of pain. Pain perception is principally a function of the lower centers of the brain; however, the upper centers and cerebral hemispheres are responsible for the interpretation of the quality of pain and other cognitive aspects of pain, which are not needed to experience pain. Perception of pain is a primitive vegetative reflex similar to thirst and hunger.

The spinal reflex is the foundational basis for pain and suffering. As long as the spinal cord is intact, the human being will experience pain and suffering. This is buttressed by the fact that patients with high cervical spinal cord injuries and transections, and quadriplegia still experience pain and suffering in the body distal to the level of the spinal cord injury. Even with

the traumatic absence of any connectivity with the upper central nervous system, the cerebral hemispheres and brainstem, a patient will continue to experience pain and suffering driven by the spinal reflex. This is in part why patients who are quadriplegic experience pain and suffering in their bodies below the levels of the spinal cord injury based upon a variety of established pathophysiological mechanisms[93-103]. Therefore, in the absence of catastrophic spinal cord and cranial injuries, a traumatized patient would experience pain and suffering.

One of the factors we consider in the determination of general and case-specific causation is analogy. Is there another disease or trauma entity that is analogous to the case in question? And in this case an analogy is that of spinal cord injuries and quadriplegia. The quadriplegic patient can still experience pain and suffering below the level of the injury, which may include but are not limited to[97]:

1. Nociceptive pain
    a. Musculoskeletal pain
    b. Visceral pain
    c. Other nociceptive pain
2. Neuropathic pain
    a. At-level spinal cord injury pain
    b. Below-level spinal cord injury pain
3. Other pain
4. Unknown pain

Gunshot wounds elicit both the fast and slow pain types. Fast pain is felt within milliseconds while slow pain is felt within about one second. Following mechanical tissue damages, biochemical tissue reactants like bradykinin, serotonin, histamine, prostaglandins, leukotrienes, potassium ions, substance P, acetylcholine, acids, and proteolytic enzymes are expressed to elicit sustained secondary biochemical pain in addition to the primary fast pain directly caused by tissue damages. The biochemical pain elicited by these biochemical reactants is a slow type of suffering pain. The intensity of pain is closely correlated with the rate of tissue damage.

The brain is responsible for and sustains consciousness in human beings. The sensation of pain induces conscious suffering since pain is a noxious sensation, which stimulates the neocortex, limbic cortex, and forebrain to cause mental pain and suffering. All these neural processes occur in 1000th's of a second [milliseconds]. The human nervous system is one of the most efficient, effective, and optimal operating systems ever known to mankind. After centuries of empirical research mankind has not been able to fully decipher and reproduce the operating systems of the human brain and nervous system.

The human brain is a post-mitotic organ and can only survive on oxygen and glucose, which are supplied by blood that come from the heart, primarily in the internal carotid arteries and the vertebral arteries. While the brain is only about 2-3% of the body weight, it receives approximately 15% of the cardiac output at a rate of 750-900 ml/min of blood. The normal range of perfusion of the brain is about 50 to 65 ml/100 g/min [80-100 ml/100g/min for the gray matter and 20—25 ml/100g/min for the white matter, at a rate of oxygen consumption of 3.5 ml/100 g/min. The normal brain tissue partial pressure of oxygen is 35 to 40 mmHg. Brain tissue oxygen levels below 30 mmHg may cause brain tissue injury, and at 20 mmHg, the risk of brain damage becomes exponentially elevated. The threshold for brain infarction is 10-12 ml/100g/min of blood supply with neuronal injury and death beginning in 60 to 180 seconds.

Being a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself. This means that when the human brain suffers any type of irreversible injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy. There are so many types of brain injuries. For the human brain to suffer irreversible global brain injury and damage, there has to be an impaired supply of oxygen and blood to the brain. The established and generally accepted median or mean reference threshold time for irreversible hypoxic-ischemic brain damage to occur is 3 to 5 minutes in cumulative time. This means that irreversible brain damage can occur in less than 3 minutes or in more than 5 minutes, but with a mean or median time of close to 3 to 5 minutes.

Pain is a basic, vegetative and primitive human reflex with a primary objective of alerting the person to remove himself from imminent danger. Given that pain is a primitive reflex, patients who are alive but are suffering a disorder of consciousness still experience pain and suffering. There is no rigid demarcation between consciousness and unconsciousness. It is a continuum or spectrum of physiological functioning, however, there are broad varying degrees of disorders of consciousness with broad varying degrees of pain and suffering physiology and biochemistry[104-107]. We cannot reasonably differentiate or quantitate the degree of pain and suffering; rather it is a qualitative question of whether a person experiences pain or not. Therefore, pain and suffering are present in all persons with disorders of consciousness and should be adequately treated[105,108-111]. In the non-communicative, unconscious patient, the most relevant aspects of response to pain are physiologic (i.e., modification in the vital parameters such as heart rate and respiration) and behavioral (i.e., modification in the facial expression, motor and visual response)[112-114].

Steffon Barber's conscious pain and suffering

Steffon Barber's conscious pain and suffering sustenance began on April 27, 2021 when the bullet perforated his scalp. At this time, Steffon Barber was conscious and aware of his surroundings. His reticular activating center was completely intact and functional. The various domains of his brain and spinal cord, and cerebral and spinal functioning were intact and perceived the noxious stimuli within 1000th's of a second. His limbic system instigated high levels of primitive adrenergic fright-flight-fight response, which caused high levels of mental, somatic and biochemical pain and suffering.

He experienced mental, somatic, and biochemical pain and suffering from every tissue destruction he suffered as a result of his ballistics injuries. He suffered severe bodily injury and severe TBI. Transfer of forensically significant kinetic energy constituted noxious stimuli which generated novel action potentials, which traveled to the spinal cord and brain to cause novel mental, somatic, and biochemical pain, and suffering.

The biochemical secondary responses of this body to the primary tissue injuries and damages precipitated biochemical, anatomic, and pathophysiological noxious stimuli, which generated action potentials within 10,000ths of a second, which were transmitted to the spinal cord and to the brain to precipitate cumulative mental, somatic and biochemical pain, and suffering. The multimodal nature of the noxious stimuli resulted in synergistic and cumulative conscious experience of very high levels of mental, somatic and biochemical pain and suffering.

Action potentials from physical or thermal noxious stimuli eventually reach the limbic system to generate mental and psychological aspects of somatic pain and suffering. The primary injuries

initiated secondary tissue injuries, systemic and tissue reactive responses, which elicited novel biochemical pain and accentuated the conscious mental and somatic pain and suffering.

Following sustenance of his trauma and serious bodily injury, he began to develop bio-physiologic deficits and sequelae of his injuries. More nerve endings in his body, soft tissues and viscerae were recruited, many more action potentials were elicited and caused increasingly higher levels of mental, somatic, and biochemical pain and suffering.

Many more types of ions, peptides, proteins, and enzymes were expressed and activated, which enhanced his biochemical pain and suffering, which synergized with his pre-existing pain and suffering to cause higher, progressive mental, somatic, and biochemical pain, and suffering.

The brain is responsible for and sustains consciousness in human beings. The region of the brain responsible for consciousness is the brainstem. The center in the brainstem, which is responsible for consciousness, is the reticular activating system, which is deeply located in the central regions of the brainstem. As long as the reticular activating system remains anatomically and electrochemically intact, an individual like Steffon Barber will remain conscious and will experience pain and suffering. Following his sustenance of severe TBI Steffon Barber's reticular activating system did not suffer any catastrophic trauma, therefore it remained intact.

As time progressed, Steffon Barber continued to experience increasingly higher levels of mental, somatic, and chemical pain and suffering due to his secondary tissue injury cascades induced by the primary traumatic injuries. His pain and suffering persisted as he received emergency medical care and was transferred to the hospital. At the hospital he received both medical and surgical treatments and was eventually discharged. Medical and surgical intervention initiated novel cascades of mental, somatic and chemical pain and suffering, which contributed to the pre-existing mental, somatic and chemical pain and suffering. His severe TBI was permanent, persisted and metamorphosed into chronic TBI and traumatic encephalopathy.

Steffon Barber manifested and suffered from and continues to manifest and suffer from the established impairments, symptoms and signs of serious bodily injury, acute severe TBI, chronic TBI and traumatic encephalopathy. He continues to experience varying modalities of conscious mental, somatic and chemical pain and suffering from the permanent, persistent, progressive and chronic TBI, the long-term sequalae of the gunshot wound he suffered. He continues to experience varying modalities of conscious mental, somatic and chemical pain and suffering from the medical and surgical interventions and treatments he received and continues to receive for this permanent brain damage and chronic TBI. The somatic symptoms and signs, motor symptoms and signs, mood symptoms and signs, behavioral symptoms and signs, cognitive and executive functioning symptoms and signs of the sequelae of his gunshot wound of the head and TBI continue to generate neurological, patho-physiological and biochemical cascades for mental, somatic and chemical pain and suffering.

The sequelae of his TBI are permanent and shall continue for the rest of his life until death. Since he was shot on April 27, 2021, Steffon Barber has suffered and experienced conscious pain

and suffering from his TBI and permanent sequelae of his TBI for 4-5 years[3,4]. He will continue to suffer and experience conscious pain and suffering from his TBI and permanent sequelae of his TBI until death[5].

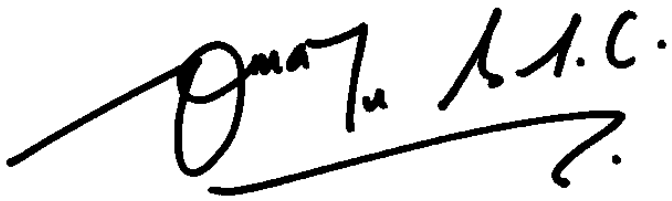
The human body continues to experience debilitating trauma-induced and physiologic biochemical pain and suffering until there is a complete cessation of all bodily functions and death as long as the spinal and cranial reflexes are intact.

I have provided my opinions and conclusions with a reasonable degree of medical and scientific certainty.

I reserve the right to amend, supplement, revise and/or modify my opinions and report, up to the time of trial, should additional information become available

Thank you.

Very truly yours,



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[3] Medicine is not an absolute science, and these estimated ranges should not be interpreted as absolute quantitative estimations of time. Quantitative ranges of any measurable index are common practice and are the standard of practice in pathology and medicine.

[4] Human events like loss of consciousness and death involve a continuum of pathophysiological events on the cellular and gross functional levels without any identifiable rigid transitions or demarcations. Therefore, the determination of the time of occurrence of these events are guided by the time the events have been reproducibly and quantifiably confirmed. For example, the time of death of any individual is determined by the time the individual was pronounced dead by a designated medical professional who has clinically assessed the patient and confirmed the patient to be dead based on prevailing, reproducible and quantifiable clinical evidence that the patient was dead.

[5] Medicine is not an absolute science, and these estimated ranges should not be interpreted as absolute quantitative estimations of time. Quantitative ranges of any measurable index are common practice and are the standard of practice in pathology and medicine.